Secondhand tobacco smoke exposure in low-income children and its association with asthma

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ABSTRACT

Secondhand tobacco smoke (SHS) is a common indoor environmental exposure that is particularly prevalent in low-income families. It has been found to be associated with asthma in some studies; however, across all relevant studies, results have been conflicting. This study aimed to determine the prevalence of SHS exposure in the home environment in a low-income, minority population and to determine the association of exposure with childhood asthma, wheeze, and oral corticosteroids use. This retrospective study analyzed self-reported data collected as part of the Kansas City Safe and Healthy Homes Partnership to determine prevalence of SHS exposure. A logistic regression model was then used to assess the association between exposure and asthma, oral steroid use, and wheeze. Overall, 40% of children lived with at least one smoker and 15% of children lived with at least one smoker who smoked inside the house. No significant association was found between asthma or oral corticosteroid use and SHS exposure. Children who lived with a smoker had a 1.54 increased odds of wheeze in the past year. A large percentage of low-income children in the Kansas City area continue to suffer the adverse effects of SHS. These data support the need for innovative public policy to protect children from such exposure in their home environment.

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Asthma is now the most common chronic condition in childhood and often leads to lifelong disability.[1] Unfortunately, the precise cause of asthma is unknown but is likely caused by many influences, including genetics, nutrition, and environmental exposures. Secondhand tobacco smoke (SHS) is one common exposure that is particularly prevalent in low-income families and has been found to be associated with asthma in some studies; however, across all relevant studies, results have been conflicting.[2]

Tobacco smoke has long been recognized for its immunosuppressive properties leading to both cancer and increased incidence of respiratory infection.[2] Recently, however, certain components of tobacco smoke were found to skew the immune response rather than having a strictly immunosuppressive effect.[3,4] These observations for the first time provided biological plausibility to the previous observed association of SHS and childhood asthma, a disease associated with uncontrolled inflammation. Because the morbidity associated with SHS is of particular concern in children who, in general, have little control over their environment but are likely to suffer the same adverse effects as the smoking adult, this potential association deserves continued investigation.

In this article, we aimed to supplement the current literature by reporting the prevalence of SHS exposure in the home environment in this low-income and predominantly minority population. We then aimed to determine the association of SHS exposure in the home with childhood asthma, wheeze, and oral corticosteroid use. We hypothesized that the odds of asthma diagnosis, wheeze, and oral corticosteroid use are all increased in children who live with smokers.

METHODS

Study Design and Aims

This retrospective analysis used data collected as part of the Kansas City Safe and Healthy Homes Partnership, which aimed to determine the impact of home remediation on asthma severity. We used a logistic regression model to assess the association between SHS exposure and asthma, oral steroid use, and wheeze controlling for confounders. Both the Kansas City Safe and Healthy Homes Partnership as well as this study were approved by the Children's Mercy Hospital Institutional Review Board.

Study Population and Data Collection

Volunteers were recruited from the greater Kansas City area as previously described.[5] Briefly, interested

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Table 1 **Prevalence of SHS exposure**

	Total $n = 308$ (%)	Low Income $n = 99$ (%)	Very Low Income $n = 207$ (%)	African American $n = 123$ (%)	Hispanic $n = 59$ (%)	White $n = 95$ (%)
No. of smokers living in the home						
0	60	64	58	54	68	65
1	24	19	27	25	27	20
2	12	14	11%	16	3	13
3+	4	3	4	4	2	2
No. who smoke inside the home						
0	85	90	83	79	98	86
1	8	4	10	13	0	6
2	5	4	6	6	1	6
3+	2	2	1	2	0	1

SHS = secondhand smoke.

volunteers responded to advertisements by contacting the study coordinator directly by phone. Inclusion criteria for participation were families with a child that has been diagnosed with asthma, chronic respiratory symptoms, chronic allergy symptoms, or other chronic symptoms affected by a home environment; were living in the Kansas City area; were staying at the same home at least 4 nights/wk; had lived in the same home for the past 6 months; planned to live in the same home for the next 12 months; and were from families with a total family income < 80% of the Kansas City median family income. Family income from the previous year was verified before enrollment.

Eligible families attended one clinic visit where written informed permission by a parent or guardian was obtained. Assent was obtained when age appropriate. A detailed questionnaire was completed that included a review of symptoms as well as medical, family, social, and environmental histories. Asthma diagnosis and history of wheeze or oral corticosteroid use in the past year were determined by parent report.

SHS exposure was determined by asking the number of individuals living with the child that smoked and, separately, the number of individuals living with the child that smoked inside the family's home.[6–10] SHS exposure was analyzed as a continuous variable of number of reported family members who smoked. Family history of asthma was defined as having at least one parent or one sibling with a history of asthma. Similarly, family history of allergy was defined as having at least one parent or one sibling with a history of allergic disease. Children from families with annual incomes <50% of the median family income for the Kansas City area were considered very low income, while others (<80% Kansas City median but >50%) were considered low income.

Statistical Analysis

All categorical variables were compared using χ^2-analysis. Continuous variables were compared using the Student's *t*-test. Logistic regression was used to predict parent-reported asthma diagnosis, history of wheeze in the past year, and history of oral corticosteroid use in the past year from number of smokers that live with the child. Age, gender, African American race, family history of asthma, family history of allergy, and income level were analyzed as potential confounders. Adjusted odds ratios and 95% CIs were estimated from the final model. All statistical analyses were performed with SAS Software, Version 9.2 (SAS Institute, Inc., Cary, NC). A value of $p < 0.05$ was considered significant.

RESULTS

Between October 2008 and November 2011, 382 families met initial screening criteria. Seventy-four families were excluded for the following reasons: 8% could not be reached beyond the initial contact, 2% lost interest in participation, 9% did not attend the clinic visit, and 1% had a change in housing. Two families were excluded because of cognitive impairment or language barrier. Three hundred eight families were included in the final analysis. *Post hoc* power analysis of the primary outcome (asthma versus no asthma) revealed that with at 38% exposure of SHS exposure in the 112 controls (nonasthma), this study had 80% power to determine a difference in odds ratio of 2.1 or greater with an $\alpha = 0.05$.

Table 1 shows the prevalence of SHS exposure in this population of children by income level and race. Overall, 40% of children lived with at least one smoker and 15% of children lived with at least one smoker who smoked inside the house. Seven percent of children lived with

Table 2 **Characteristics of compared groups**

	Asthma $n = 196$ (%)	No Asthma $n = 112 >$ (%)	Wheeze $n = 223$ (%)	No Wheeze $n = 85$ (%)	OCS $n = 160$ (%)	No OCS $n = 148$ (%)
Demographics						
Gender (female)	76 (39%)	53 (47%)	87 (39%)	42 (49%)	64 (40%)	65 (44%)
Age, yr (mean ±SD)	8.3 ± 4.1#	†7.1 ± 4.6	7.9 ± 4.3	7.7 ± 4.4	7.4 ± 4.0	8.3 ± 4.6
Race/ethnicity						
African American	84 (43%)	39 (35%)	94 (42%)	29 (34%)	72 (45%)§	51 (34%)§
Hispanic	36 (18%)	23 (21%)	39 (17%)	20 (24%)	21 (13%)§	38 (26%)§
Caucasian	55 (28%)	40 (36%)	65 (29%)	30 (35%)	47 (29%)§	48 (32%)§
Other	21 (11%)	10 (9%)	25 (11%)	6 (7%)	20 (12%)§	11 (8%)§
Family history of asthma	†94 (48%)	†27 (24%)	95 (43%)	26 (31%)	77 (48%)§	44 (30%)§
Family history of allergy	52 (27%)	27 (24%)	64 (29%)*	15 (18%)*	46 (29%)	33 (22%)
Socioeconomic status						
Low income	66 (34%)	33 (30%)	74 (33%)	25 (29%)	102 (64%)	105 (71%)
Very low income	129 (66%)	78 (70%)	147 (67)	60 (71%)	57 (36%)	42 (29%)
Lives with at least one smoker	79 (40%)	43 (38%)	96 (43%)*	26 (31%)*	65 (41%)	57 (39%)
SHS exposure in the home	28 (14%)	17 (15%)	35 (16%)	10 (12%)	22 (14%)	23 (16%)

*$p < 0.05$.
#$p < 0.05$.
§$p < 0.05$.
OCS = oral corticosteroid; SHS = second hand smoke.

more than one person who smoked inside the house in which they lived. The prevalence of SHS exposure was similar in both income groups and across races.

Table 2 shows the demographics of each of the analyzed groups. A significant difference was found in mean age as well as family history of asthma between the asthma and no asthma groups. A significant difference was found in family history of allergy between the wheeze and no wheeze groups. A significant difference was also found in race and family history of asthma between the oral corticosteroid group and no oral corticosteroid group. No other differences were seen between the compared groups.

Logistic regression was used to predict asthma associated with SHS exposure after adjusting for both age and family history of asthma. Similarly, a logistic regression model was used to predict wheeze associated with SHS exposure adjusting for family history of allergy and, finally, to predict oral corticosteroid use in the last year associated with SHS exposure after adjusting for race and family history of asthma. Table 3 shows the odds ratios and 95% CI and adjusted odds ratios and 95% CI estimated from the model. No significant association was found between asthma or oral corticosteroid use and SHS exposure before or after adjusting for confounders. Children who lived with a smoker, however, were found to have a 1.54 increase odds of wheeze in the past year for each smoker who lived in the house (95% CI, 1.09, 2.18) after adjusting for family history of allergy. Similar models were performed where the explanatory variable was living with a smoker who smoked indoors as well as maternal smoking only; however, no significant difference was found, and the results are not reported here.

Table 3 **Odds ratios estimated from logistic regression**

	OR	95% CI	AOR	95% CI
Asthma*	1.26	0.96, 1.66	1.21	0.91, 1.60
Wheeze#	1.55	1.10, 2.19	1.54	1.09, 2.18
OCS§	1.13	0.89, 1.44	1.07	0.83, 1.37

**Adjusted for age and family history of asthma.*
#Adjusted for family history of allergy.
§Adjusted for ethnicity and family history of asthma.
OR = odds ratio; AOR = adjusted odds ratio; OCS = oral corticosteroid.

DISCUSSION

This study indicates that despite efforts to reduce SHS exposure with indoor smoking bans in public spaces, a large percentage of low-income children in the Kansas City area continue to suffer the adverse effects of SHS. We found 40% of children live with a smoker and 15% are regularly exposed to SHS in their home environment. In addition, we found that living with a smoker was associated with a history of wheeze in the past year; however, this study failed to reach significance when

analyzing the association between living with a smoker and childhood asthma or oral corticosteroid use.

The nationwide prevalence of home SHS exposure in children is unclear but has likely declined over the previous decades. In 2006, Pirkel *et al.* described a decline of detectable cotinine in the National Health and Nutrition Examination Survey population from 88% in the late 1980s to 43% in 2001–2002.[11] Because indoor smoking bans were rare in 2001–2002, this estimate includes children exposed to SHS in public spaces as well as their home environment. Thus, although this estimate is similar to that found in this study, the overall prevalence of SHS in the home is likely higher in our population because exposure in public spaces is not included. Furthermore, the true prevalence of home SHS in our population is likely even higher than reported, because our study relied on parent report, which is likely to underestimate true exposure.[12] More recently, in 2011, Oberg *et al.* estimated that 24% of children and nonsmoking adults in the United States, Canada, and Cuba were exposed to SHS; and 40% of children and nonsmoking adults worldwide were exposed using 2004 data.[13] Our data indicate a higher proportion than this estimate, which is consistent with a known higher burden of exposure in low-income populations. Finally, a 2013 report found that 17% of asthmatic patients are exposed to SHS in their home, a proportion similar to that in this report.[14]

In 2006, the Surgeon General reported on the adverse health effects of involuntary exposure to SHS and included a detailed meta-analysis, which found an adjusted pooled odds ratio for wheeze of 1.25 for children who had at least one parent who smoked, an observation that is further supported by these data.[2] The same report also concluded that a causal relationship between parental smoking and prevalent asthma can be inferred. This statement contradicts the findings of this study; however, the adjusted pooled odds ratio for this meta-analysis was too small for detection in this study. Furthermore, the Surgeon General's report went on to conclude that "evidence is suggestive but not sufficient to infer a causal relationship between SHS exposure from parental smoking and the onset of childhood asthma," implying that the relationship is complex and not yet completely defined.

The strength of this study is the unique population that participated, because it investigated the prevalence of home SHS exposure in low-income children from the Kansas City area. In addition, ~40% of the studied population is African American, a population of children that is particularly susceptible to the development of asthma. The primary weakness of this study is the small population size. Because of the small sample size, this study was not sufficiently powered to detect a small difference in odds for the primary outcome group (asthma versus no asthma). In addition, we acknowledge that this study is subject to misclassification bias because the compared groups were classified by parent report only. Although parent report of both asthma diagnosis and asthma medication use is thought to be valid for epidemiological research, parent-reported wheeze often differs from that of physician diagnosis. Parent-reported wheeze may therefore be more indicative of upper respiratory illness rather than actual wheeze.[15–17]

In summary, these results further support the need for innovative public policy to protect children from SHS exposure in their home environment, an environment in which they are likely to have little control. Although this study provided further compelling evidence of an association between wheeze and living with a smoker, additional studies are needed to clarify the association of SHS exposure in the home and childhood asthma.

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